Effects of Repeated Valsalva Maneuver Straining on Cardiac and Vasoconstrictive Baroreflex Responses

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Introduction: We hypothesized that repeated respiratory straining maneuvers (repeated SM) designed to elevate arterial BPs (arterial baroreceptor loading) would acutely increase baroreflex responses. ***Methods:*** We tested this hypothesis by measuring cardiac baroreflex responses to carotid baroreceptor stimulation (neck pressures), and changes in heart rate and diastolic BP after reductions in BP induced by a 15-s Valsalva maneuver in 10 female and 10 male subjects at 1, 3, 6, and 24 h after performing repeated SM. Baroreflex responses were also measured in each subject at 1, 3, 6, and 24 h at the same time on a separate day without repeated SM (control) in a randomized, counterbalanced cross-over experimental design. ***Results:*** There was no statistical difference in carotid-cardiac and peripheral vascular baroreflex responses measured across time following repeated SM compared with the control condition. Integrated cardiac baroreflex response (ΔHR/ΔSBP) measured during performance of a Valsalva maneuver was increased by approximately 50% to 1.1 $\pm$ 0.2 bpm $\cdot$ mm Hg^{-1} at 1 h and 1.0 $\pm$ 0.1 bpm $\cdot$ mm Hg^{-1} at 3 h following repeated SM compared with the control condition (0.7 $\pm$ 0.1 bpm $\cdot$ mm Hg^{-1} at both 1 and 3 h, respectively). However, integrated cardiac baroreflex response after repeated SM returned to control levels at 6 and 24 h after training. These responses did not differ between men and women. ***Conclusions:*** Our results are consistent with the notion that arterial baroreceptor loading induced by repeated SM increased aortic, but not carotid, cardiac baroreflex responses for as long as 3 h after repeated SM. We conclude that repeated SM increases cardiac baroreflex responsiveness which may provide patients, astronauts, and high-performance aircraft pilots with protection from development of orthostatic hypotension.

Keywords: Valsalva maneuver, heart rate, vasoconstriction, baroreceptors.

THE IMPORTANCE of arterial baroreflexes in the regulation of BP is underscored by the development of severe hypotension under conditions of sinoaortic denervation in dogs (16). Similarly, human subjects who exhibit attenuated carotid-cardiac baroreflex function have more frequent episodes of severe hypotension and intolerance when exposed to experimentally induced orthostatic challenges (6–8,11,12,18–20,26,27). Likewise, the significance of baroreflex control of vascular resistance in the regulation of BP during orthostatic challenge has been demonstrated by the association of attenuated peripheral vasoconstriction with development of orthostatic hypotension and intolerance (3,13,14,20,21,34). Therefore, if baroreflexes can be acutely 'trained' to increase the cardiac and/or peripheral vascular responses to reductions in arterial BP, it might be possible to ameliorate or eliminate compromising hypotension that can develop in individuals with central hypovolemia caused by aerospace and military operational environments.

There is evidence to suggest that loading baroreceptors by periodic elevation of arterial BP may provide a primary stimulus for maintenance or enhancement of cardiac and vascular baroreflex response. Consistent with this hypothesis is the observation that elevated pulse pressures in isolated carotid sinuses of dogs sensitized baroreceptor afferent firing (4,5). Elevated arterial BPs, i.e., arterial baroreceptor loading, is the normal response to standing upright in terrestrial gravity (7). In contrast to the attenuating effect of exposure to environments of relative low gravity and physical inactivity (6,7,11,12,20), cardiac responses and peripheral vasoconstrictive reserve to baroreceptor stimulation are enhanced in individuals exposed to high gravity (9) and acute maximal exercise (10,17–20). It is therefore conceivable that aortic or carotid baroreflexes become less sensitive in the absence of regular transient elevations in BP that are caused by the response to routine exposure to the upright posture and/or physical activity.

We previously hypothesized that development of effective treatments for orthostatic hypotension might include techniques to maintain or increase baroreflex sensitivity prior to reambulation (7). Although acute exercise designed to elicit maximal arterial pulse pressure elevations has proven effective in reversing orthostatic hypotension and intolerance in subjects exposed to prolonged periods of bedrest and wheelchair confinement (19,20), most patients who have been confined

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for prolonged periods of inactivity as a result of trauma injury or debilitating disease are incapable of performing intense physical exercise. Likewise, performing physical exercise is not always possible in aerospace or military operational environments. Therefore, the use of a more clinically applicable technique of loading baroreceptors is necessary. In a previous experiment (9), we observed increased cardiac and cardiopulmonary baroreflex responses in subjects who performed respiratory straining maneuvers during high-G acceleration training. In this experiment, we tested the use of a series of repeated respiratory straining maneuvers (repeated SM) similar to those used by high-performance aircraft pilots to determine if and for how long baroreflex sensitivity can be acutely increased. We hypothesized that repeated SM designed to elevate arterial BPs (arterial baroreceptor loading) would acutely increase baroreflex responses. If the use of this procedure proved successful in elevating baroreflex responses, this technique could represent a countermeasure in clinical and aerospace settings for acute treatment of orthostatic hypotension as well as be applied for assessment and prediction of baroreflex function in patients, pilots, and astronauts.

METHODS

Subjects: Twenty healthy, normotensive, non-smoking men (n = 10) and women (n = 10) served as subjects. The descriptive data for the female subjects were (mean ± 1 SEM): age, 31 ± 3 yr; height, 170 ± 2 cm; weight, 68.5 ± 3.1 kg; heart rate, 64 ± 4 bpm; systolic BP, 111 ± 3 mm Hg; diastolic BP, 71 ± 3 mm Hg; and mean BP, 84 ± 1 mm Hg. The descriptive data for the male subjects were (mean ± 1 SEM): age, 32 ± 3 yr; height, 178 ± 2 cm; weight, 79.3 ± 4.0 kg; heart rate, 64 ± 3 bpm; systolic BP, 129 ± 2 mm Hg; diastolic BP, 76 ± 2 mm Hg; and mean BP, 94 ± 1 mm Hg. The subjects were not aircraft pilots nor had they undergone any particular type of exercise training. Individuals taking prescription drugs were excluded from the study. Because of the potential effects on baroreflex function, subjects refrained from exercise and stimulants such as caffeine and other non-prescription drugs 48 h prior to testing. However, we were unable to dictate menstrual cycle phase of female subjects during scheduling for baroreflex testing. During an orientation period that preceded each experiment, all subjects were made familiar with the laboratory, the protocol, and the procedures. Experimental procedures and protocols were reviewed and approved by the Research Council and Human Use Committee of the U.S. Army Institute of Surgical Research and the Human Investigative Review Board of the Kennedy Space Center for the use of human subjects. Each subject gave written informed voluntary consent to participate in the experiments.

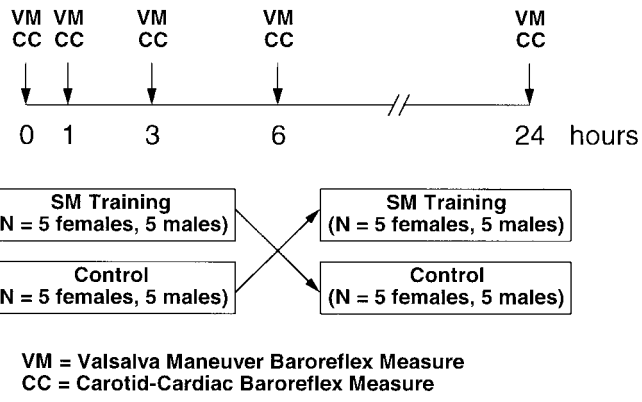

Fig. 1. Experimental protocol. See text for detailed description.

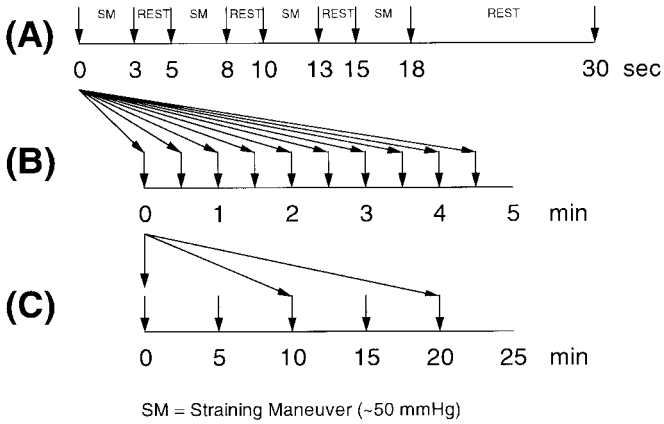

Fig. 2. Respiratory straining maneuver (SM) protocol. See text for detailed description.

Protocol: A diagram of the experimental protocol is presented in **Fig. 1**. Each subject completed two testing sessions that entailed various measures of their stimulus-response relationships of cardiac and vascular baroreflexes: (a) once after a series of repeated straining maneuvers (repeated SM), and (b) once in the control (no repeated SM) condition. The order of treatment was randomized and counterbalanced so that five men and five women underwent baroreflex testing after repeated SM first, and five men and five women underwent baroreflex testing without repeated SM (control condition) first. A minimum of 6 d (mean ± SE = 7.2 ± 0.3 d) intervened between the two experimental test sessions. Subjects maintained their normal sleep pattern during the 2 wk of testing. Both experimental protocols were initiated at the same time of day.

Repeated SM Sessions: Following measurements of baseline baroreflex responses conducted prior to time 0 (Fig. 1), repeated SM was initiated. Each subject performed a regimen of repeated SM in the sitting posture designed to elevate intrathoracic (arterial) BP by approximately 50 mm Hg on each maneuver in a manner similar to that observed in high-performance aircraft pilots (30). For each SM, subjects blew into a mouthpiece connected by a short plastic tube to a calibrated pressure gauge. A diagram of the repeated SM session is presented in **Fig. 2**. One 30-s cycle of repeated SM consisted of three 3-s SMs each followed by 2-s non-strain interval (rest), with a fourth 3-s SM followed by a 12-s non-strain interval (Fig. 2A). Each 30-s cycle of repeated SM was repeated 10 times consecutively (total of 5 min) followed by a 5-min rest period (Fig. 2B). After 5 min of rest, each subject performed a second 5-min repeated SM session followed by 5 min of rest and a third 5-min repeated SM session (Fig. 2C). Therefore, the total repeated SM session was completed in 25 min.

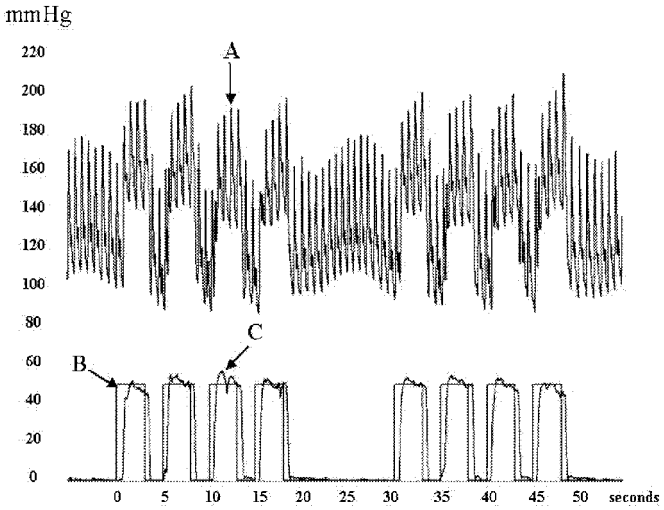

Fig. 3. Recordings of arterial BP (A), target level (50 mm Hg) for expiratory pressure (B), and actual expiratory pressure (C) generated by a subject during repeated SM. This recording represents two consecutive 30-s repeated SM segments.

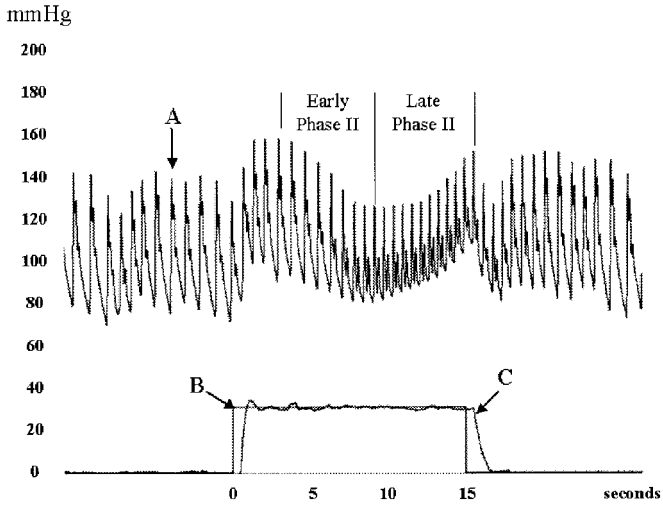

Fig. 4. Recordings of arterial BP (A), target level (30 mm Hg) for expiratory pressure (B), and actual expiratory pressure (C) generated by a subject during performance of a standard Valsalva maneuver.

During each 5-min repeated SM period, beat-to-beat arterial pressure was estimated with a non-invasive Finapres infrared finger plethysmographic device with the hand placed at heart level and expiratory pressures were measured continuously with a calibrated pressure transducer (**Fig. 3**). Baroreflex function tests were repeated at 1, 3, 6, and 24 h after the completion of the repeated SM session (Fig. 1, time 0). Subjects were instructed to control their physical activity to sitting and minimal walking during data collection for both the SM and control treatments. With the exception of the repeated SM, the same protocol was followed on the control experimental day.

Valsalva maneuver: Each test battery for baroreflex function included measurement of heart rate and BP responses to Valsalva maneuvers followed by the measurement of heart period response to neck pressure stimuli. Subjects performed three Valsalva maneuvers at a controlled expiratory pressure (30 mm Hg) in the seated posture. Each trial included a 30-s baseline period followed by a 15-s strain period, and a 2-min post-strain recovery period. For Valsalva strains, subjects blew into a mouthpiece connected by a short plastic tube to a calibrated pressure gauge. Heart rate, arterial pressures, and expiratory pressures were measured continuously. Beat-to-beat arterial pressure was estimated with a non-invasive Finapres infrared finger plethysmographic device. Excellent estimates of directly measured intra-arterial pressures during Valsalva maneuvers have been demonstrated with this device (23). Heart rate and BP responses from three trials were averaged in a phase-by-phase manner for early phase II and late phase II (**Fig. 4**). The ratio $\Delta HR/\Delta SBP$ was used in the analyses for early phase II because of its usefulness in describing integrated cardiac baroreflex responsiveness (31,32). For late phase II, ΔDBP was used in the analyses as a marker for sensitivity of baroreflex-mediated control of peripheral vascular resistance since α-adrenergic blockade with phentolamine results in abolished or marked reduction of the late phase II response (31).

Measurement of carotid-cardiac baroreflex: Carotid baroreceptor-cardiac reflex responses were measured in the supine posture with the experimental method described previously (25). A diagram of the pressure profile, electrocardiogram recording, and plot of the stimulus-response relationship between R-R interval and estimated carotid distending pressure is presented in **Fig. 5**. Briefly, a stepping-motor driven bellows was used to deliver a series of pressure and suction steps to a Silastic neck chamber. During held expiration, a pressure of about 40 mm Hg was delivered to the chamber

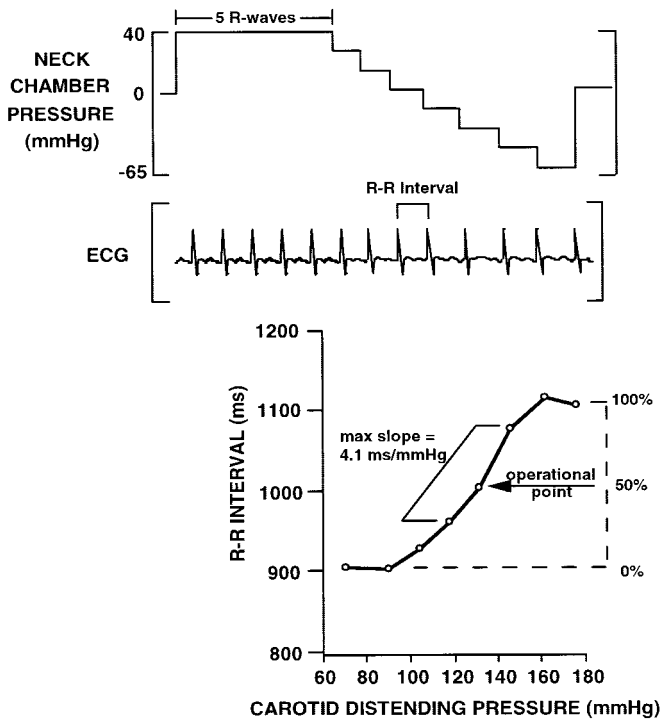

Fig. 5. Quantification of carotid-cardiac baroreflex responsiveness. See text for detailed description.

and held for 5 R-waves; then, with the next R-wave, the pressure was sequentially stepped to about 25, 10, −5, −20, −35, −50, and −65 mm Hg, and then returned to ambient pressure. Pressure steps were triggered by R-waves so that neck chamber pressure changes were superimposed on naturally occurring carotid pulses. During each test session the stimulus sequence was repeated five times and data averaged for each subject. Previous studies in our laboratory indicated that baroreceptor stimulus-sinus node response relationships measured in this way are highly reproducible. R-R intervals for each pressure step were plotted against estimated carotid distending pressures (CDP = systolic pressure minus neck chamber pressure applied during the heart beat). From the average of each five-trial sequence of responses, the stimulus-response baroreflex relationships were reduced to maximum slope (determined by application of least squares linear regression analysis to every set of three consecutive points on the stimulus-response relationship), and operational point (determined from the calculation [(R-R intervals at 0 mm Hg neck pressure – minimum R-R intervals)/(maximum R-R interval – minimum R-R intervals)] × 100%). The maximum slope provided an index of reflex sensitivity and the operational point provided a measure of the relative baroreflex buffering capacity for pressures above and below resting levels.

Statistical analysis: The statistical analysis was a standard two group (male, female) by two treatment (repeated SM, control) by four time periods (1, 3, 6, and 24 h) mixed model analysis of variance. The model was mixed in the sense that subjects were nested within gender groups and crossed with treatments and time [i.e., one between subjects factor (gender) and two within subjects factors (treatment and time)]. All main effects and subsequent interactions were analyzed across three dependent effects (carotid-cardiac baroreflex sensitivity, ΔHR/ΔSBP response during early phase II of the Valsalva maneuver, and ΔDBP response during late phase II of the Valsalva maneuver). Precise p-values were calculated for each independent effect and reflect the probability of obtaining the observed or greater effect given only random departure from the assumption of no effects. For the main effect of time, and interactions involving time, p-values were adjusted with the Huynh and Feldt correction to account for possible non-random error structure related to time (22). Standard errors (without removal of between-subject variation) presented in the table and text, and depicted in the figures represent raw measures of variation specific to the specified subgroup rather than variability specific to the factors being tested or the variability associated with statistical tests and subsequent p-values.

RESULTS

Descriptive data: Average and standard error baseline values for age, height, weight, heart rate, and BPs for women and men are presented in the Methods section of this paper. Male and female groups were matched for age. Women demonstrated similar baseline heart rates compared with men, but had lower baseline arterial BPs, body weight, and height. Since cardiac and vascular baroreflex responses in male and female subjects were not statistically different (see the following statistical analyses results), graphical data were presented as a combined group of 20 subjects.

Repeated SM profiles: Mean (±SE) heart rate increased from 73 ± 2 bpm at baseline to 80 ± 2 bpm during SM and returned to 73 ± 2 bpm during the 2-s interval between SMs. An example of the arterial loading effect, i.e., elevated arterial BP, during each SM is demonstrated in Fig. 3. Mean (±SE) values for baseline systolic, diastolic, and mean arterial BPs were 137 ± 4, 80 ± 3, and 99 ± 3 mm Hg, respectively. SM increased baseline systolic, diastolic, and mean arterial BPs to 161 ± 4, 103 ± 3, and 122 ± 3 mm Hg, respectively. During the 2-s interval between SMs, systolic, diastolic, and mean arterial BPs recovered to baseline levels, i.e., 137 ± 4, 81 ± 2, and 100 ± 3 mm Hg, respectively. Throughout baseline, straining maneuvers, and 2-s recovery, pulse pressure was unchanged (56 ± 2, 58 ± 2, and 55 ± 2 mm Hg, respectively).

Cardiac baroreflex response to Valsalva maneuver: The treatment by time mean (±SE) values for ΔHR/ΔSBP response during early phase II of the Valsalva maneuver is presented in **Fig. 6** (upper panel). There was a large main treatment effect of repeated SM ($F(1,18) = 10.43$, $p = 0.0046$). The average ΔHR/ΔSBP was 1. 0 bpm · mm Hg^{-1} with repeated SM compared with a control value of 0.7 bpm · mm Hg^{-1}. Although repeated SM consistently produced a larger heart rate elevation to reduced BP during early phase II of the Valsalva maneuver irrespective of both gender and time, there was a slight interaction of repeated SM with time, suggesting that the repeated SM effect was diminishing as time increased ($F(3,54) = 3.29$, $p = 0.0421$). No other main effects or interactions were statistically discernible ($F \leq 1.86$, $p \geq 0.1893$).

Vascular baroreflex response to Valsalva maneuver: The treatment by time mean (±SE) values for ΔDBP response during late phase II of the Valsalva maneuver are presented in Fig. 6 (lower panel). Across both treatment and time, ΔDBP response in women was consistently higher than men ($F(1,18) = 6.0114$, $p = 0.0247$). Overall, average female ΔDBP response was 19.2 mm Hg compared with an average male response of 13.4 mm Hg. Since there was no consistent pattern in the difference between treatment means over time, no main effects (other than gender) or interactions were statistically discernible ($F \leq 1.17$, $p \geq 0.3301$).

Carotid-cardiac baroreflex: Since gender did not influence carotid-cardiac baroreflex responses across time during either repeated SM or control experimental conditions, the data were combined and analyzed as a sample size of 20. **Fig. 7** demonstrates the mean (±SE) values for the treatment by time for the maximum slope (upper panel) and operational point (lower panel) of the carotid-cardiac baroreflex. No statistical main effects or interactions were found for either maximum slope ($F \leq 1.55$, $p \geq 0.2296$) or operational point ($F \leq 1.91$, $p \geq 0.1432$).

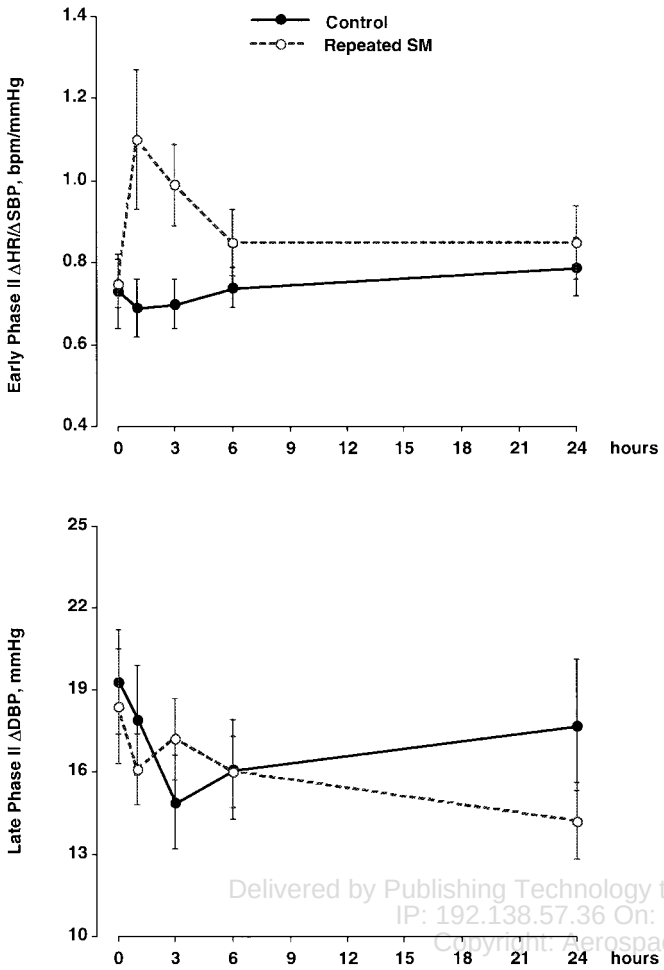

Fig. 6. Responses in ΔHR/ΔSBP during early phase II (top panel) and in ΔDBP during late phase II (bottom panel) to the Valsalva maneuver at baseline (0 h) and at 1, 3, 6, and 24 h after repeated SM (open circles, broken line) and without repeated SM (closed circles, solid lines). Values are mean ± standard error.

DISCUSSION

There is compelling evidence that arterial baroreceptor loading by periodic elevation of arterial BP may provide a primary stimulus for maintenance or enhancement of cardiac and vascular baroreflex response (4,5,7,9). We therefore hypothesized that a series of repeated respiratory straining maneuvers (repeated SM) designed to elevate arterial BPs (arterial baroreceptor loading) would acutely increase baroreflex responses. Repeated SM increased the ΔHR/ΔSBP (integrated cardiac baroreflex) response during early phase II of the Valsalva maneuver, an effect that was maximal at 1 to 3 h after repeated SM, but diminished with time and returned to baseline by 24 h. Therefore, our findings support the hypothesis that repeated SM acutely increased an integrated cardiac baroreflex response. However, since repeated SM failed to affect the ΔDBP response during late phase II of the Valsalva maneuver, our results do not support our hypothesis that baroreflex control of peripheral vascular resistance was affected by acute arterial baroreceptor loading.

Repeated SM did not affect the operational point and heart period response to carotid baroreceptor stimulation. Therefore, these results do not support the hypothesis that the specific arterial baroreceptor loading induced by our protocol of repeated SM was capable of increasing the isolated carotid-cardiac baroreflex response (Fig. 7, top panel). The lack of impact of repeated SM on carotid-cardiac baroreflex function may not be surprising since acute maximal resistive exercise which produces elevations in arterial pressure loading similar to that of our repeated SM protocol, i.e., elevated arterial BP without increased pulse pressure (29), also failed to increase isolated carotid-cardiac baroreflex responsiveness (33). In contrast, the isolated carotid-cardiac baroreflex sensitivity was acutely increased for as long as 24 h after performance of dynamic cycle ergometer exercise (10,17–20), which produces both elevations in mean arterial pressure and pulse pressure (29). Since there may be conditions at all BPs when baroreceptors are stimulated more by a change in pulsatile pressure rather than a change in mean arterial pressure (1), our observations in the present investigation give rise to the notion that increased pulse pressure rather than simply elevating arterial pressure may be necessary to provide an adequate stimulus for increasing the responsiveness of the carotid-cardiac baroreflex.

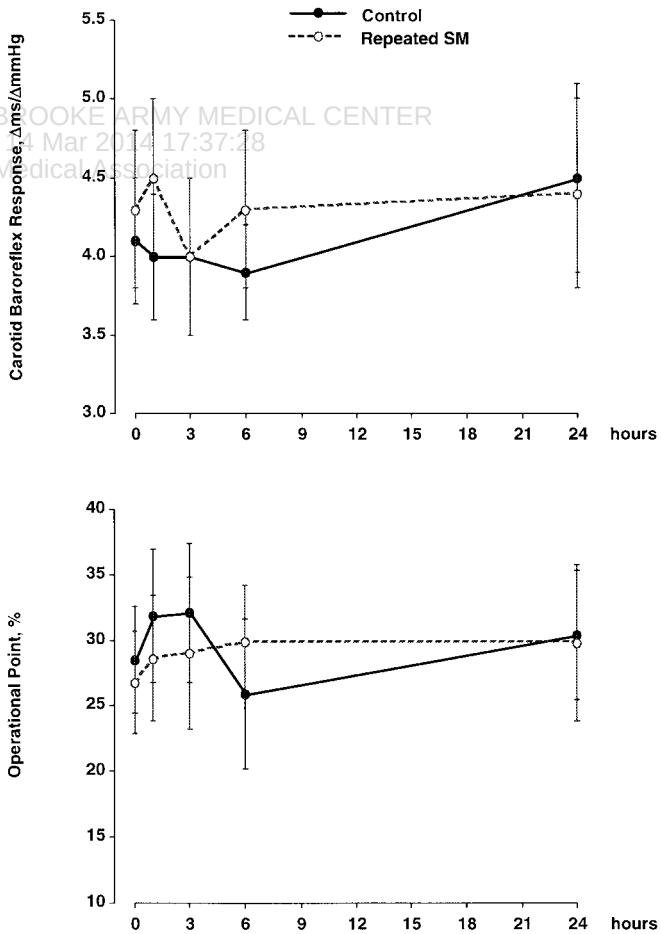

Fig. 7. Maximum slope (gain) (top panel) and operational point (bottom panel) of the carotid-baroreflex response at baseline (0 h) and at 1, 3, 6, and 24 h after repeated SM (open circles, broken line) and without repeated SM (closed circles, solid lines). Values are mean ± standard error.

Future experiments are necessary to test this intriguing hypothesis.

The elevation in heart rate response measured for an equal drop in BP during the early phase II of the Valsalva maneuver represents an integrated reflex cardiac response to arterial hypotension (i.e., involves stimulation of both aortic and carotid baroreceptors). Our findings that the cardiac response during the early phase II of the Valsalva maneuver was increased at 1 and 3 h after repeated SM while the isolated carotid-cardiac response was unaltered suggests that the specific arterial loading stimulus (i.e., increased systolic, diastolic, and mean arterial pressure without change in pulse pressure) had a specific effect on aortic, but not carotid, baroreflex response. If the carotid baroreceptors proved to be more responsive to arterial loading patterns that specifically increase pulse pressure, the results of this investigation may be the first to suggest that optimal maintenance of cardiac baroreflex responses requires transient elevations in both arterial and pulse pressures.

Assessment of the operational impact of the increase in aortic-cardiac baroreflex response with repeated SM is difficult since our experiment did not include tests of clinical hypotension. However, calculations performed on a hypothetical orthostatic response and results of the present investigation may place these baroreflex adaptations in perspective. In the present study, the average $\Delta HR/\Delta SBP$ was 0.4 and 0.3 bpm $\cdot$ mm Hg^{-1} greater at 1 and 3 h after repeated SM compared with the control condition. During an acute hypotensive event where SBP is reduced from 120 to 100 mm Hg ($\Delta SBP = 20$ mm Hg), the increased cardiac baroreflex response caused by repeated SM would translate to an elevation in heart rate of 6 to 8 bpm greater than in the control condition. Given a reduction in stroke volume to 50 ml during this hypothetical orthostatic stress, repeated SM would contribute to a 0.3 to 0.4 L $\cdot$ min^{-1} higher cardiac output. These calculations suggest that the increased cardiac baroreflex response associated with repeated SM in the present investigation could provide protection against the onset of hypotension by ameliorating the reduction in cardiac output and arterial BP during conditions of central hypovolemia.

We recently reported that individuals who undergo repeated exposure to high-G acceleration (G training) in a simulation of high-performance aircraft maneuvers demonstrated greater isolated carotid-cardiac and cardiopulmonary-vascular baroreflex responses compared with non-trained control subjects (9). In order to provide protection against G-induced loss of consciousness during their training sessions, G-trained subjects were required to perform straining maneuvers similar to those performed by our subjects in the present investigation. In the absence of pre-training measures (9), it was not possible to determine if differences in baroreflex function between G-trained subjects and non-trained controls resulted from exposure to high-G acceleration, from straining maneuvers, or a combination of both. Despite an increased heart rate reflex response during Valsalva maneuvers at 1 and 3 h post repeated SM, the results of the present investigation provide the first evidence that repeated anti-G straining maneuvers do not significantly alter long-term (>24 h) carotid-cardiac, aortic-cardiac, and cardiopulmonary-vascular baroreflex responses. Therefore, it is clear that accentuated cardiac and vascular baroreflex responses reported 24 h after exposure to high-G acceleration (9) cannot be explained by straining maneuvers. However, we cannot dismiss the possibility that repeated SM effects conducted over prolonged periods (e.g., weeks, months) may contribute to increased baroreflex functions.

In the present study, the isolated carotid-cardiac baroreflex response in women (4.2 $\pm$ 0.9 msec $\cdot$ mm Hg^{-1}) was similar to that in men (3.7 $\pm$ 0.6 ms $\cdot$ mm Hg^{-1}), and was unaltered by repeated SM for both gender groups. We observed the same pattern for the reflex heart rate response in women (-0.8 ± 0.2 bpm $\cdot$ mm Hg^{-1}) and men (-0.7 ± 0.1 bpm $\cdot$ mm Hg^{-1}) during the Valsalva maneuver. The population of female subjects who participated in the present investigation differed from those in previous investigations where cardiac baroreflex responses to arterial baroreceptor stimulation (2,8,24) and orthostatic hypotension (15) were attenuated in women compared with men. We have no clear explanation for the presence of gender differences in cardiac baroreflex response in previous investigations (2,8,24) but not in the present investigation except for random probability of subject selection. In contrast to cardiac baroreflex responses, female subjects in the present study demonstrated an exaggerated peripheral vascular reflex response (as indicated by a 43% greater ΔDBP during late phase II Valsalva maneuver) compared with male subjects. Since we were unable to dictate the time of testing with regard to our female subjects' menstrual cycles, we have no way of assessing whether the gender difference in peripheral vascular reflex response to the Valsalva maneuver was influenced by the menstrual cycle. However, the difference in peripheral vascular reflex response to the Valsalva maneuver between our male and female subjects in the present investigation was similar to our previous observations when all women were measured in the follicular phase of their menstrual cycle (8,28). Thus, it seems unlikely that gender similarities or differences observed in the present investigation were influenced by menstrual phase. More importantly, independent of gender similarities or differences in baroreflex functions, the training effect for increased sensitivity of the aortic-cardiac baroreflex was independent of gender. Therefore, we conclude that our repeated SM protocol is equally effective in women as in men.

As with any investigation, the present experiment is not without limitations. The order of Valsalva and carotid-cardiac baroreflex tests was not counterbalanced across subjects. Therefore, it is possible that performing a Valsalva straining maneuver for assessment of integrated baroreflex function may have systematically affected the measurement of the carotid-cardiac baroreflex response. However, previous data from our laboratory (10) demonstrated a baseline carotid-cardiac baroreflex sensitivity (approx. 4 ms $\cdot$ mm Hg^{-1}) that was not altered at 3, 6, and 24 h, virtually identical to the results observed in the present investigation. These comparisons suggest that it was unlikely that the Val-

salva straining maneuver test had any significant effect on the measurement of the carotid-cardiac baroreflex response. More importantly, our randomized counterbalanced order for application of independent variables (i.e., control vs. repeated SM straining treatments) allows us to validly conclude that there was no effect of repeated SM straining on these baroreflex responses.

Implications for Application to Aerospace and Military Medicine

Since attenuated arterial baroreflex control of cardiac responses is associated with orthostatic compromise (6–8,11,12,14–16,18–20), the results of this investigation are relevant to military personnel in their recovery following confinement to bedrest as a result of combat or training injury, during exposure to environment conditions that compromise normal orthostatic performance (e.g., standing at attention, heat, dehydration, aircraft maneuvers), or during hemorrhage. For instance, incompetence of BP regulation appears to be a significant issue for military operations. More than 10,000 cases of unexplained syncope were reported during fiscal year 1998 across all branches of the DoD (U.S. Army Medical Education Department, Fort Sam Houston, TX). Repeated short duration (3 s) Valsalva maneuvers (i.e., straining maneuver training) performed by soldiers prior to field tasks could reduce the incidence of unexplained syncope in the military. Similarly, repeated SM performed by astronauts within 3 h of reentry to earth gravity from a space mission could reduce the incidence of postflight orthostatic intolerance. Repeated SM could be used by high-performance aircraft pilots prior to a sortie to provide enhanced baroreflex function that might optimize BP maintenance during aerial combat maneuvers.

In summary, we demonstrated that performance of repeated respiratory straining maneuvers similar to those performed by high-performance aircraft pilots (i.e., repeated SM) can increase the baroreflex-mediated elevation in heart rate induced through aortic baroreceptor stimulation by systemic hypotension. This training effect might provide some protection against the development of hypotension and compromise to operational performance and well being typically experienced by personnel in aerospace and military environments.

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